Society Proceedings.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, February 1st, 1887.

The President, C. L. DANA, M.D., *in the Chair.*

DR. C. L. DANA reported a case of

PACHYMENINGITIS HÆMORRHAGICA,

with large meningeal hemorrhage pressing chiefly on leg centre; right hemiplegia; total paralysis in leg; aphasia; hemianæsthesia; convulsions limited to arm and face; death.—Exhibition of specimen.

The patient was a woman about 68 years old, and came into the hospital with complete motor aphasia, and unable to give any previous history. She had no paralysis at first, but three days after admission she had a general convulsion, followed by right hemiplegia, total in the leg, and some right-sided anæsthesia. On the second and third days, she had a series of brief localized convulsions, involving the face bilaterally and the right arm. These were carefully observed. The movements were clonic, beginning in the muscles of the lower jaw. The other peculiarities were these: 1. The pupils remained small during the convulsions. When wider convulsive centres are discharging, as in general epileptic convulsions, the pupils are dilated. It is not probable that in this case there was some uræmic element, because the post-mortem disclosed a sufficient cause for them. 2. The conjugate deviation of the eyes was at first, and very temporarily, toward the side of the lesion, and away from the paralyzed side. The head also was turned toward this side. When this occurs, it is ordinarily spoken of as a paralytic deviation. This does not explain it here, since almost immediately the head and eyes were turned strongly to the opposite and paralyzed side.

The speaker suggested that the first deviation is due to an inhibition of the activity of the associated nuclei of the third and sixth nerves that innervate the external and internal recti of the two eyes. There are many facts which tend to show that the first stage of convulsion is a transient paralysis due to a sudden discharge of inhibition centres. These are of a higher, more developed class than the centres for motor discharges, and would be affected first. We would have then loss of consciousness, inhibition of motion, and muscular relaxation ; then motor discharges and tonic and clonic convulsions.

3. The temperature on the paralyzed side was one degree higher than normal, and higher by a degree than that of the other side. This is the rule in intra-cerebral hemorrhage and hemiplegia, but the speaker was not aware that it has been established in cortical hemiplegias. In meningeal hemorrhages, the temperature is often below normal, according to Minot. 4. The presence of hemianæsthesia.

The patient died on the third day. Post-mortem showed chronic pacchymeningitis over both convexities, but more on the left side. On the left convexity there was a very extensive fresh meningeal clot pressing upon and flattening especially the upper half of the central convolutions. Brain substance normal.

CORTICAL EPILEPSY WITH TEMPORARY APHASIA. SYPHILITIC GUMMA COMPRESSING THE LEFT SECOND FRONTAL CONVOLUTION IN ITS LOWER POSTERIOR PART. RECOVERY.

Dr. M. Allen Starr related the history of the case. Chas. S., aged 32, had always been healthy and a hard worker. He had an attack of sciatica four years ago, and three years ago had a hard chancre. He had never had convulsions or nervous affection. Family history good. During November, 1886, and the first two weeks of December he did not sleep well, was slightly dizzy, his head ached a good deal, chiefly at night. December 15th, while walking with a pail in his right hand, he suddenly let it drop, losing all power in the hand and arm. There was numbness in the hand. He was unable to speak to his companions. He did not feel dizzy or notice any pain in the head; he did not lose consciousness nor fall. He understood his friends' questions, but could not answer. Power in the hand and arm and speech returned within half an hour. The next morning he went to work as well as usual. Two days later a second attack occurred, beginning with a numb feeling in the tips of the fingers, gradually extending up the hand and arm. Then the fingers became rather forcibly flexed and stiff, but by a voluntary effort he could straighten them. No clonic spasms of the fingers, and wrist and elbow were not bent. The numbness and stiff feeling soon extended to the face, which was drawn to the right side with some force. Speech was again lost. No loss of consciousness. The attack lasted about twenty minutes. Such attacks had occurred every other day, then every day, and finally twice a day up to January 3d, and during this time the headache and insomnia were increasing steadily. The character of the attacks was not uniform. Sometimes the spasm would begin in the face, though usually the arm was first affected. Both were involved in every attack, but the spasm and numbness never reached the leg. The hand felt cold during the attack, though warm to the touch. On one occasion he had for four days great difficulty in making himself understood by words. Examination by Dr. Starr showed slight paresis, and slight tactile anæsthesia in right hand. No affection of face or speech. No cardiac symptoms, though suffering from headache. Percussion of skull did not reveal any tender spots. Thrombosis, endarteritis syphilitica, diffuse encephalitis with sclerosis were excluded, and the diagnosis was reached of gumma in the membranes, resting upon the brain surface, giving rise to irritation and consequently to an occasional nervous discharge, but not of sufficient size to cause any destruction. Location of tumor was equally clear, the relative situations of the cortical centres for the arm, face, and for the movements of speech in the lower two-thirds of the anterior central convolution and in the posterior part of the third frontal convolutions were likened to a reversed L. All these centres were irritated during the attacks, the irritation sometimes beginning in one, sometimes in another. If the tumor pressed upon the lowest posterior part of the second frontal convolution which would lie inside of the L, an irritation radiating from it might reach all three centres equally. The total intermission of the local symptoms might be explained by such a location, since no symptoms were known to occur from injury of this part. The fact that numbness in the hand and face uniformly accompanied the attacks of spasm seemed to indicate that the areas for these parts coincide with the motor areas.

Another point of interest was the distinctly motor character of the aphasia.

The treatment ordered was first inunction of mercury, and secondly iodide of potash daily in divided doses. He had one attack two days after beginning treatment, but since that time he had

had no return of the symptoms. Iodide of potash was still being taken.

DISCUSSION ON DR. STARR'S PAPER.

Dr. Seguin had seen several cases whose symptoms resembled those of the case recorded in Dr. Starr's paper. The prognosis of even non-syphilitic cortical lesions with this symptomatology was not absolutely unfavorable. One of the cases to which he referred was that of a Cuban who came to his clinic about nine years ago. He had never had syphilis, yet he described epileptic attacks of the true cortical kind, such as have been obtained by experiments upon animals during the last few years. The hand would become numb, and then the seat of a vibratory sensation; finally contraction would occur in the hand, when the face, and almost simultaneously the leg would be affected, and he would lose consciousness. According to his friends' account, general convulsions then occurred. He had had quite a number of these seizures, yet examination showed no anæsthesia, no affection of the optic nerve, and, so far as the speaker could recollect, no motor impairment. The patient had received a preparation composed mostly of the bromide of potassium, to which a little of the iodide was added. He improved immediately, and four years ago the attacks ceased. Once in a while he has the sensation of wires in the hand, and the hand becomes stiff, but the face is never affected. The case was a beautiful illustration of the localization of a lesion in the centres for the hand, the discharge radiating to those of the face and leg of the same side, then to the opposite side, with loss of consciousness.

The patient had also been the object of the bracelet experiment. He was a powerful man, and had exerted great force, arresting many attacks in this way. The speaker was satisfied that syphilis was absent, while the amount of the iodide was too small to explain a cure upon the ground of a syphilitic affection.

Dr. Starr had been much interested in the case which Dr. Seguin had related. He had recently had a case of unilateral convulsions in his office. The patient was a small boy. The attack commenced in the eyes and face. The eyes turned to the right, then the head turned to the right, then the arm, then the leg became affected. During the attack, the speaker had asked suddenly, "What is your name?" The boy promptly replied, "Arthur," and then relapsed into the convulsion. He supposed that the reply was reflex, as the boy was unconscious at the time and did not afterward remember the occurrence. He would like the opinion of the members upon the point.

DR. DANA asked what Dr. Seguin had considered the lesion in his case.

DR. SEGUIN had never ventured to surmise beyond the fact that there was a nerve lesion, and that there was no syphilis in the case.

DR. STARR asked Dr. Seguin whether in localized convulsions numbness were not the rule.

DR. SEGUIN replied that it was, but he did not know that the reason was yet sufficiently established, although Von Monakow had associated anæsthesia with lesions of the motor zone.

DR. SHAW referred to a case seen first four years ago. While at work as a jeweler, the patient fell off his bench in a convulsion. The face and the left arm were convulsed; the leg was not affected. Sometimes only the side of the face was affected. He had seen many of these attacks limited to the side of the face in his office. The patient complained of numbness in the arm and the side of the face, and the speaker felt sure that the tactile sensibility was not as good upon that as upon the other side. The patient denied syphilis. Upon ophthalmoscopic examination the nerves were found pale, and the visual field restricted in its upper part. There was no change until about six months ago when, without the loss of vision, he was found to have choked disk. This had gone on to atrophy, and the man was now blind. There was no paralysis. From the choked disk, of course, the speaker had now diagnosed a tumor, but he referred to the case on account of the anæsthesia and the spasms, and their resemblance to those in Dr. Starr's case.

DR. SACHS referred to the case of a man who some years ago while working upon the Capitol at Albany, had fallen some distance, was found unconscious, but recovered. A few weeks later he developed symptoms which alarmed his friends, and he had now some of the physical and nearly all of the mental signs of general paresis: the irregular pupils, the facial tremor, the tremor of the tongue, and the deteriorated mentality. The speaker referred to the case because of the traumatic incident, and because every three or four weeks this man had an attack of numbness beginning in the fingers and creeping up the right arm to the face. There never were convulsions, but both the patient and his wife, who is a very intelligent person, claim that paralysis follows upon these attacks of numbness. After three or four hours, both the paresis and the numbness disappear, and he has a very severe headache, lasting one or two days. The speaker thought there was a question of chronic meningitis with encephalitis possibly in this case. It was evidently a cortical affair.

DR. DANA thought that cortical epilepsy might develop, like idiopathic epilepsy, without an appreciable lesion. He recalled a case, that of a young man who was kicked in the front of the thigh by a horse. Twitching of the leg developed, similar to that of cortical epilepsy. Thrilling and numbness of the arm and face followed. In a year true hemiepileptic attacks, during which he lost consciousness, developed, and upon giving him ether for stretching the nerve, he went into the status epilepticus There was no history of syphilis. Apparently cortical epilepsy was developed just as true idiopathic epilepsy in other cases.

DR. E. C. SEGUIN read a paper entitled,[1]

A CONTRIBUTION TO THE PATHOLOGY OF THE CEREBELLUM.

DISCUSSION OF DR. SEGUIN'S PAPER.

DR. BRADNER spoke by invitation. He had been the attendant in the case of the child to which Dr. Seguin had referred. He had not prescribed the washing out. It had been done by a prominent physician of the place. He had done it a number of times during three years, but had then refused, believing that the child had brain disease of some form. He saw the patient first in November. The vomiting was always in the morning. There was no pain connected with it then, although a frontal headache had developed during the last few months. The treatment prescribed by Dr. Seguin had been the iodide of potash fifteen grains t. i. d., increasing five grains daily until 100 grains were taken at a dose. The course had been interrupted by several attacks of acute gastritis, but the child had had those attacks previously—they did not appear to depend upon the medication. The eyesight has been perfect ; the child could detect the smallest point made by a lead pencil or needle. While using the iodide his headache had improved, as had some other symptoms, but he retained his old man's gait.

DR. SEGUIN remarked that, though seeing well, the child had typical choked disk.

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[1] See this JOURNAL, p. 217.

Dr. Bradner added that since seeing Dr. Seguin he had obtained a history of injury in the case. Two years ago, and just before the commencement of his illness, he had, while trying to skate, fallen and received a severe blow on the back of his head. One result of this injury had been abiding terror at the sight of a body of water or ice.

Dr. Shaw had shown a child at the American Neurological Association in 1878, on account of a peculiar ataxic gait like that of locomotor ataxia. The later symptoms had pointed to a tumor of the cerebellum.

Dr. Putnam Jacobi referred to the recent collection of cases by Bernhardt. All cases published previous to 1878 had been collected by Nothnagel. Dr. Seguin's cases tended to confirm Nothnagel's laws. There were many resemblances between the symptoms of cerebellar tumor and those of tumor in other parts of the brain. The peculiar violence of the headache and the choked disk found in most cases of cerebellar disease might, she thought, be due to the increased pressure of a tumor confined by the tentorium. This element had not been commented upon, and she would like Dr. Seguin's opinion upon it.

Another point referred to the fact that the laws formulated by Nothnagel recognize the possibility of complete latency of the tumor, no symptoms at all being present when but one lobe of the cerebellum is affected; such symptoms appearing only when the tumor encroaches upon the central lobe. In one of Dr. Seguin's cases the tumor only occupied the lateral lobe. Of course, an indirect affection of the central lobe might be present even in such a case.

Dr. Leszinsky referred to a case to which he was called in consultation by Dr. Alexander. The boy had the typical gait of spastic paraplegia; any attempt to stand caused spastic contractures in the limbs. The ankle clonus was present, and the knee-jerk was exaggerated. There were no cerebral symptoms; the fundus was normal in both eyes. The father and mother were both alcoholic and unworthy people. Finally, the child became unable to walk, but there were still no cerebral signs, no vomiting. Two months before death the fundus was still normal. Later, tremor developed, and paralysis of the abducens, of the fifth, and of the third nerves. Total blindness occurred. The nurse, a graduate of Bellevue, was positive that the child could see nothing. It died in a convulsion. Unfortunately, the body was immediately frozen, and the specimen was unfit for sections. The tumor was found occupying one side of the cerebellum. The spinal cord was not fit for examination. There was a well-developed meningitis, and the paralysis was accounted for by considerable exudation about the cerebral nerves. One peculiarity had been a subnormal temperature during one stage of the case.

DR. STARR had had occasion some time ago to make a collection of cases of cortical lesions from American literature. He had at the same time made a collection of cases of cerebellar disease from the same sources. As the data obtained only corroborated Nothnagel's results, he never published them. But Dr. Seguin having remarked upon the value of corroborative evidence, he would briefly refer to them now. From 1860 to 1884, one hundred and sixty cases of cerebellar disease were reported in American literature. In only 40 of these were the symptoms and the autopsies described with sufficient accuracy to warrant conclusions. These 40 the speaker had quite thoroughly analyzed. In 4 there were no symptoms; in 1 of these there was congenital atrophy of the cerebellum; in 2 abscesses; and in 1 a large cyst. Of the remaining 36 presenting symptoms, there was headache in 36; inco-ordination in 25; vertigo in 20; vomiting in 18; blindness in 14; dim vision in 6; diplopia or strabismus in 7; deafness in 7; facial spasm or paresis in 4; hemiplegia in 9; general paralysis in 4; mental symptoms in 8; stupid 7; mania 1; convulsions in 7; sexual desire increased in 2. Males 23, females 17. Ages, between 1 and 20 years, 11 cases; between 20 and 40 years, 16 cases; between 40 and 60 years, 9 cases; over 60 years, 1 case; age not stated in 3.

In two of the eleven cases in which inco-ordination did not occur, the lesion probably involved the middle lobe. In cases where inco-ordination occurred, various parts of the cerebellum were involved, but the probability was that the middle lobe was affected in the majority. There were only two instances of increased sexual desire. Bernhardt had found but one instance in ninety cases of cerebellar tumor, and Nothnagel but two cases. The speaker thought that it might be thrown out as a symptom of cerebellar disease, and regarded as one of accidental occurrence. The escape from vertigo in Dr. Seguin's case was explained, perhaps, by the recent discoveries in the anatomy of the course of the acoustic nerve. This nerve served for the sense of hearing and the sense of space. The centre for hearing is in the pons. Edinger finds the centre for equilibrium in the cerebellum, to which acoustic fibres pass by way of the middle peduncle. From this centre the central tract probably passes onward to the superior peduncle. Dr. Seguin says that the superior peduncle escaped in his case. It is therefore natural that vertigo should have not occurred.

The tendency to rotation was an interesting feature in these cases. There was a tendency to fall or turn forward in two cases: the lesion in both was a tumor in the vermis, in the anterior part. There was a tendency to fall to the right in two cases, in one there being a tumor in the left middle peduncle, and in the other an abscess in the same part. There was a tendency to fall to the left in two cases, in one there being an abscess in the right middle peduncle, and in the other a tumor in the left middle peduncle. A patient of Nothnagel, whom the speaker had seen in Vienna, when getting up in bed had always a tendency to turn to the right side. Nothnagel considered this due to vertigo. It was only present when the patient was erect. The patient felt as though about to fall to the left side, and hence turned to the right. This case had a tubercle of the left middle peduncle of the cerebellum. Nothnagel considered this symptom only produced by affection of the middle peduncle. In the two cases in the table where the tendency was to turn to the right, there was disease of the left middle peduncle; while in the two in which the tendency was to the left, in one the right peduncle, and in the other the left peduncle was affected. No rule can, therefore, as yet be laid down as to the cause of this symptom.

Dr. Seguin closed the discussion. He was not surprised to learn of the little boy's fall, as he was a firm believer in the traumatic origin of these conditions. It was very difficult to obtain a history of fall. He could not give an opinion upon the question propounded by Dr. Putnam Jacobi. In regard to ascribing the vertigo to the acoustic nerve, he was not yet certain that the acoustic nerve had cerebellar origin. He thought it would be difficult to trace fibres through the lateral peduncle of the cerebellum, the vermis, and the anterior peduncle.

He said Dr. Starr had probably made a mistake when he referred to Nothnagel connecting rotation with disease of the middle peduncle. Middle vermis he had probably meant to say.

Dr. Starr was aware that Nothnagel connected incoördination with disease of the vermis, but rotation towards one side was considered by Nothnagel as a symptom of disease in one or the other middle peduncle passing from the pons to the cerebellum.